Trauma Rounds

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Shock

EDWARD VERRIER, MD:* The case we wish to discuss today is that of a 69-year-old man who was involved in a high speed automobile accident and was admitted to San Francisco General Hospital shortly after injury.

On initial assessment in the trauma room, the patient was comatose. Blood pressure was 70/0 mm of mercury and there was pronounced peripheral vasoconstriction. An endotracheal tube was immediately placed and large bore cutdowns were established in the antecubital fossa and at the ankle. After volume resuscitation with two liters of Ringer's lactate solution, vital signs stabilized at near normal levels permitting diagnostic assessment to be carried out.

No evidence of external head trauma was found but there was extensive bleeding from a facial laceration below the right eye. The pupils were bilaterally fixed with minimal light response. On examination of the chest, rib fractures were palpable on the right; however, breath sounds were equal in both hemithoraces. Findings on examination of the abdomen were unremarkable, but because of lack of consciousness and evidence of body trauma, peritoneal lavage was carried out; there was a bloody return. A compound fracture of the right leg was noted to be present. Urine was clear on analysis. The hematocrit reading was 35 percent.

On x-ray films no skull fracture was seen; however, anterior dislocation of the C-1 cervical spine was noted. Fractures of ribs 7 and 8 on the right were confirmed by x-ray studies of the chest. Findings on an intravenous pyelogram showed good excretion bilaterally. A cystogram was not done. On x-ray films of the long bones, a right tibial-fibular fracture was seen. During x-ray examination, the patient's blood pressure again dropped and he was taken immediately to the operating room. Administration of prophylactic antibiotics was begun shortly following induction of anesthesia.

On exploratory laparotomy, two units of whole blood—from a ruptured spleen—was present in the peritoneal cavity. Splenectomy was carried out. Concomitantly, facial fractures were reduced and bleeding from maxillofacial injuries controlled. Despite administration of six units of blood, hypotension occurred once again. Tube thoracostomy was done and 600 ml of whole blood was noted in the right side of the chest. After this, bilateral exploratory burr holes were carried out and brain contusion was shown to be present, but no epidural or subdural hematoma. The compound right leg fracture was debrided and stabilized in plaster. A total of 17 units of whole blood was required by the time surgical procedures were completed.

The postoperative course was stormy, charac-

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terized by coma for the first four days. This cleared somewhat over the next week and seemed to respond to steroid administration. Another major problem was respiratory insufficiency. Positive pressure ventilation and 40 percent oxygen were required initially to maintain the patient's arterial partial pressure of oxygen (Po_2) at 60. This deteriorated on day five as manifested by increasing pulmonary infiltrates and deteriorating pulmonary function despite positive end expiratory pressure (PEEP). Pseudomonas was cultured from the trachea. Despite treatment with gentamycin, progressive pulmonary deterioration occurred. Overt sepsis developed, with positive blood cultures and a fall in peripheral perfusion and urinary output. Despite optimal fluid replacement and ionotropic cardiac support the patient died on the eighth postoperative day.

DONALD TRUNKEY, MD:* *This case represents all the problems manifested by a patient resuscitated from major injury and advanced shock. We are privileged today to have Dr. Robert Freeark who is Professor and Chairman of the Department of Surgery at Loyola University and Dr. John Davis who is Professor and Chairman of Surgery at the University of Vermont to discuss this particular case. I would like to ask Dr. Freeark to open the discussion and ask him to direct his remarks to hypovolemic shock.*

ROBERT FREEARK, MD:† This patient initially exemplified the most common cause of shock in trauma patients, a reduction of circulating volume. Although in this patient there were multiple sources of bleeding we must remember that there are other sources of volume loss in severe shock. The principal of these is sequestration of fluid in the intracellular space. Third space loss into bowel and body cavities contributes to the hypovolemic state. Therefore, the first thing that must be done in resuscitation of hypovolemic shock is volume replacement.

One must be aware, however, of other possible causes of shock in trauma patients—particularly those causes related to the heart, such as myocardial contusion or tamponade. Another possibility is tension pneumothorax from a combination of lung laceration and positive pressure ventilation. These are associated with high central venous pressures, as opposed to shock due to hypovolemia. Therefore, it is imperative to look at the neck veins. If they are full or a central venous pressure measurement is high, the cause of shock is cardiogenic or pulmonary.

With these few exceptions, after the described venous assessment one can assume that most shock in the trauma patient is hypovolemic. Immediate fluid replacement should be begun by placing large-bore cutdowns and starting infusion of Ringer's lactate solution. At the same time, a specimen of blood should be sent for cross matching and an ample supply of blood obtained. We infuse the first two to three bottles of Ringer's lactate solution just as fast as possible—that is, within 5 to 10 minutes. The sodium load is of no consequence as long as the patient is in shock. Adequate resuscitation is determined by a good urine output from a bladder catheter—that is at least a ml of urine per minute in an average adult. Other useful signs include good peripheral perfusion and improvement in sensorium.

There is considerable speculation regarding the nature of organ failure following hypovolemic shock. It has been my observation that with prompt correction of volume deficits, the incidence of such problems as respiratory failure of this type results from secondary complication of sepsis which occurred in this patient.

DR. TRUNKEY: *What is the role of overhydration in respiratory distress?*

DR. FREEARK: As I indicated earlier, I believe that prompt resuscitation with Ringer's lactate solution is optimal therapy. I do not believe the fluid therapy causes the lung lesion. I agree with Dr. Blaisdell's concept that the advanced shock lesion is a lesion consisting of diffuse increase in the vascular permeability. These leaky lung vessels result in the "wet lung" seen following shock. Failure to restore adequate volume results in persistent shock aggravating the leak. Overhydration may increase lung edema but this type of edema is readily reversible, while the former is not.

F. WILLIAM BLAISDELL, MD:‡ *Dr. Freeark, in view of the subsequent outcome in this case, in which prophylactic antibiotics were administered because of compound fracture, would you comment on the use of antibiotics in hypovolemic shock?*

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DR. FREEARK: I do not know all the details in this case, but certainly respiratory distress syndrome and sepsis developed. Since the organism was pseudomonas it is safe to speculate that the use of prophylactic antibotics may have set the stage for this opportunistic infection. To my knowledge there is no evidence that the use of antibiotics, when started early, will prevent the adult respiratory distress syndrome, particularly when they are used in a shotgun or nonspecific manner. I do not have a strong opinion on the use of antibiotics, but would use them for specific clinical indications such as compound fracture or grossly contaminated wounds. I would start administration as early as possible—preferably in the Emergency Room.

DR. BLAISDELL: *What about the use of steroids in hemorrhagic shock? Do they stabilize cell membranes and prevent further problems?*

DR. FREEARK: There is no convincing evidence that the use of steroids in hypovolemic or cardiogenic shock have any salutory effect. I also think we have enough evidence now that steroids may do harm by depressing the immune mechanism. I am not even convinced that they should be used in head trauma. I believe that they probably did more harm than good in this particular instance.

DR. TRUNKEY: *Let's move on to the second problem in this case, septic shock, and ask Dr. Davis to discuss this.*

JOHN DAVIS, MD:* The patient in this case had been severely injured and there had been two instances of hypotension, one initially and one intraoperatively. This in addition to such other factors as major soft tissue injury is probably why sepsis developed and the patient died. Other contributing factors would include the endotracheal intubation, the chest injury, the multiple transfusions and the surgical operation. He may have aspirated with his initial injury. It is not unusual for pseudomonas to present as it did in this case. Pseudomonas is a saprophytic organism that causes little in the way of clinical problems until there is tissue damage or shock. Then it seems to be able to overcome host defenses and obtain a foothold. The use of prophylactic antibiotics such as the cephalosporins seems to increase its incidence relative to other organisms. With contamination of the airway, a rapidly spreading bronchopneumonia then develops and the patient's condition deteriorates over a period of 24 to 48 hours.

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DR. TRUNKEY: *We certainly agree with that concept and we also feel that the steroids may play a very important role since in the injured patient there probably is an immunodeficiency that is just added to by the steroids.*

DR. DAVIS: As you know this is a controversial point and when sepsis develops we prefer to use steroids, particularly in large doses. The theoretical benefits would include a reduction of the capillary membrane leak and lysosomal breakdown and there may be an ionotropic effect on the heart. I am not sure, however, that use of steroids would have made a difference in this case, or that their clinical benefit has been established beyond question. You do occasionally see a patient, however, in whom there is response to the use of steroids, at least temporarily, and for that reason I continue to use them.

DR. BLAISDELL: *Dr. Davis, would you give us a definition of septic shock? Do you believe there is such an entity?*

DR. DAVIS: Yes, I think septic shock is a distinct entity. Most shock is hypovolemic or cardiogenic. Septic shock is decreased nutritive perfusion that may actually develop in the face of increased cardiac output, suggesting that there may be arteriovenous shunting. It is not a simple volume defect and it is not a simple pump defect. It may be a combination of both. There are certainly peripheral vascular effects secondary to sepsis that produce diffuse vascular permeability with loss of vascular volume into the interstitial tissues. There may well be certain "toxic" problems that occur because of septic invasion of tissues. These toxins are characteristic of Gram-negative infection but in some instances may also be associated with Gram-positive infections. Hypotension ultimately develops, with inadequate perfusion as manifest by lactic acidosis. Decreased peripheral perfusion develops and terminally cardiac depression occurs due to the "toxic" effects of advanced infection.

DR. BLAISDELL: *Dr. Davis, would you comment on the specific aspects of treatment of septic shock?*

DR. DAVIS: Treatment is threefold: (1) remove the source of infection, (2) correct the volume

deficit and (3) support the heart. The first is often easier said than done, particularly in cases such as the one presented today where the problem was diffuse. Blood, urine, wound and sputum smears or cultures, or both, will define the organism and direct specific antibiotic therapy. Abscesses or other sources of infection that can be treated by surgical drainage should be looked for and treated promptly and definitively. Gallium scans now provide a means of locating intraperitoneal infections so as to permit simple direct drainage of intraperitoneal collections or pus.

Patients with sepsis require vigorous fluid therapy so as to maintain urine output. Central venous pressures or left-sided cardiac pressures should be monitored using a Swan-Ganz catheter. High pressures associated with poor perfusion should be treated with cardiotonic drugs. These may include isoproterenol (Isuprel®), dopamine or steroids depending upon your clinical experience and the response of the individual patient.

DR. BLAISDELL: *Dr. Freeark, do you have any comments to add to the concept of septic shock?*

DR. FREEARK: I would agree with Dr. Davis. I am sure that septic shock has a direct effect on the heart and would like to emphasize the importance of support of the myocardium during sepsis. I also think we have to keep in mind that when sepsis involves a large surface it may produce a severe volume deficit and large amounts of volume replacement may be required for proper resuscitation.

DR. BLAISDELL: *Dr. Davis, do you feel that severe trauma or shock depresses the immune system?*

DR. DAVIS: That is a difficult question. If one were to take a number of patients such as the one discussed today, resuscitate them and treat them in the same way, some would survive and some would die. I do not know if there is an immune defect in those that die. I think there is evidence that shock and trauma are additive and do in some way increase susceptibility to infection. In the patient discussed today there were two episodes of hypovolemic shock and both may have contributed to an immune deficiency.

FRANK LEWIS, MD:* I would like to second Dr. Freeark's recommendation against using steroids in the presence of an injury to the head. For some years the British did not use steroids following head trauma, believing them to be of no value. It has been my impression working in the intensive care unit that in patients with head injury and multiple trauma treated with steroids there is a special risk of their conditions suddenly deteriorating because of sepsis. In these patients standard treatment also tends to be less effective and a deterioration of condition may occur in a much shorter time than in patients not so treated.

DR. FREEARK: I would like to mention one other complication of steroid therapy: the problem of peptic ulcer. I recall a patient who died because of this complication while recovering from head injury. Because of this I have followed the literature very carefully and I can find no convincing evidence that administration of steroids is beneficial in traumatic brain injury.

DR. BLAISDELL: I would like to thank our two discussants today—Drs. Freeark and Davis. We have been honored to have them visit us. They have made a number of important points, which I will summarize. The primary treatment of hypovolemic shock is volume replacement. Volume replacement must be sufficient to correct blood volume losses and also third space loss due to vascular permeability changes, losses into the cells and sequestration of fluid in the gut and body cavities. Severe hypovolemic shock seems to render the patient vulnerable to infection probably via depression of immune defenses so that sepsis is not prevented by prophylactic antibiotics. In fact, administration of antibiotics seems, if anything, to increase the incidence of virulent infections such as pseudomonas. Septic shock results from a combination of cardiac depression and volume loss due to an increase in vascular permeability generated by toxins liberated by the infecting organisms. Nonnutritive blood flow or arteriovenous shunting aggravates the circulating deficit. Treatment consists of (1) eradicating the infection by surgical drainage or specific antibiotic therapy, (2) establishing adequate volume and (3) myocardial support.

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